vessels of lesser calibre. Pressure applied high on the sternum near the sternal angle can cause fracture of the body of the sternum, and pressure applied directly to the ribs may result in costal fractures. Force applied over the xiphoid process, epigastrium or lower thorax is the likely cause of hepatic or splenic injuries. The increased negative intrathoracic pressure which is created between compressions by the resultant expansion of the intact thorax is presumably the reason for bone marrow embolization from fracture sites to the pulmonary arteries.[4]

Prevention of complications from external cardiac massage can only be obtained by the application of appropriate pressure at the proper anatomical site—the inferior portion of the body of the sternum. As described by Kouwenhoven and Jude,[5] appropriate pressure is that amount necessary to move the sternum 3 or 4 cm. toward the vertebral column. It is emphasized that the force sufficient to do this is influenced by two separate factors: the anatomical construction of the patient's chest and the strength of the person employing the procedure.

Slight pressure with one or two fingers will suffice in a newborn infant[4] but in an adult, whose chest is less resilient, pressure with both hands in such a way that the pressure is transmitted through the heel of one, usually are required. The application of total body weight, recommended by some local heart association bulletins,[6] can be avoided in the majority of cases, this extreme being necessary only when the thorax is extremely rigid. Undoubtedly, optimal use of external cardiac massage would include the least application of force at the proper site and the smallest excursion necessary to provide palpable pulsations in the carotid, femoral or brachial arteries that are accompanied by a reduction of the pupillary dilatation that accompanies cardiac asystole.[6]

The previous editorial comment,[3] "A heavy hand on a frail chest is not without risk," might well be restated as, "A heavy hand improperly placed on a frail chest invites injury."

SUMMARY

In a case in which external cardiac massage was carried out, many complications reported in the literature occurred—multiple rib fractures, fracture of the sternum, hemothorax, hemopericardium, lacerations of the liver and rupture of the inferior vena cava near its junction with the right atrium. In addition a previously unmentioned complication, laceration of pulmonary parenchyma, was observed at autopsy.

The causes and prevention of complications from this procedure are discussed.

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Jejunogastric Intussusception Following Subtotal Gastric Resection

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RETROGRADE JEJUNOGASTRIC INTUSSUSCEPTION is a rare complication of gastroenterostomy or subtotal gastric resection. Irons[5] reported the one hundredth case in the world literature in 1955, and a total of 26 cases reported in this country to 1959 were reviewed by Salem,[7] who added a case of his own. In excellent reviews of this subject by Mason[6] and Caudell[3] it was pointed out that none of the reporting observers had seen more than two patients with this disease, and that the literature is made up almost exclusively of single case reports. The serious nature of this form of high intestinal obstruction with its uniformly fatal ending if untreated, and the almost universal delay in diagnosing this little-known condition prompts this single case report.

The clinical features of jejunogastric intussusception are classical, and the diagnosis, if thought of, can be made quickly and confirmed easily. In the cases reviewed by Foster[4] he noted that the mortality was five times as high when operation was delayed beyond 48 hours from the onset of symptoms as it was when correction was carried out earlier. Unfortunately, the diagnosis has been established at autopsy in almost 30 per cent of the reported cases.

The earlier cases followed gastroenterostomy, but (understandably) in the more recent cases the precedent operation was gastric resection. Caudell[3] attempted to settle the question of who first described this disease by naming it the Bozzi-Delfino-Steber syndrome. However, we agree with Caudell that an eponym is unnecessary for this medical rarity, and that the descriptive term jejunogastric intussusception has gained popular acceptance.

Shackman[8] classified jejunogastric intussusception into three types: 1, Intussusception of the afferent loop; 2, Intussusception of the efferent loop (the most common type); and, 3, a combined afferent and efferent loop prolapse. Van Prohaska[9] pointed out that only the type 2 efferent loop pro-

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lapse through the gastrojejunal stoma has been observed in association with subtotal gastric resection. To be more precise, this entity should be called a jejuno-jejunogastric intussusception, for this is the anatomical arrangement as actually seen at operation.[5]

There is a chronic recurrent type of jejunogastric intussusception, apparently a nebulous form of this disease, which is usually diagnosed as postgastrectomy syndrome, marginal ulcer, or dumping syndrome, and sometimes it is ascribed to neurosis. The true cause of the recurrent epigastric discomfort, nausea and vomiting finally becomes apparent when a barium study is made during a period of active symptoms.

The features of acute, fulminating jejunogastric intussusception were demonstrated in a patient recently seen by us.

REPORT OF A CASE

A 56-year-old Caucasian man was admitted May 11, 1962, at 7:30 p.m., with a history of the sudden onset an hour previously, of severe pain with waves of colic in the upper abdomen. Vomiting soon followed. The pain was localized and of such severity that the patient "broke out in a cold sweat." The vomitus had the appearance of coffee grounds at first, but quickly became obviously sanguinous.

Five years previously, the patient had had a duodenal ulcer treated by subtotal gastric resection. From time to time afterward he complained of nausea, but not of pain.

On admittance the patient appeared pale and acutely ill. Blood pressure was 180/96 mm. of mercury, the pulse rate 60 and respirations 20 per minute. Oral temperature was 96.4° F. The abdomen was flat and soft, tender on deep palpation in the left epigastrium, but no mass was noted. Peristalsis was slightly hypoactive. No tenderness was elicited by rectal examination.

The hemoglobin content was 12.2 gm. per 100 cc. of blood and leukocytes numbered 7,000 per cu. mm. with 66 per cent neutrophiles. Results of urinalysis and serum amylase determination were within normal limits. X-ray films of the abdomen disclosed no free air.

Following admission, the patient vomited 200 cc. of dark bloody fluid, and an additional 200 cc. of lighter, sanguinous gastric content was aspirated with a Levin tube. Before entering the hospital the patient had been given 100 mg. of meperidine (Demerol®) intramuscularly, and another 75 mg. was administered a half hour after admission, but he continued to complain of pain and was obviously in great distress.

A diagnosis of acute jejunogastric intussusception was made and confirmed by an emergency barium study (Figures 1 and 2).

Operation was carried out five hours after admission, with the patient under spinal anesthesia. An efferent loop type of jejuno-jejunogastric intussusception was found as shown in Figure 3. It was quickly determined that tugging on the distal jejunum would tear the bowel, whereas pressure on the intussusceptum through the wall of the stomach resulted in the reduction of 30 cm. of edematous jejunum which was viable, but showed early vascular changes of strangulation. The previous operation had been a subtotal gastric resection with an antecolic, antiperistaltic, Polya gastrojejunostomy. The jejunum opposite the stoma was dilated three times normal size. No attempt was made to fix the efferent loop to prevent a recurrence of the prolapse.

The convalescence was uneventful, and the patient was discharged on the fifth postoperative day.

DISCUSSION

The etiology of jejunogastric intussusception is conjectural, but there is no question about the mechanical features as observed at operation. Unlike some forms of isoperistaltic intussusception seen in the ileocecal area, never in retrograde jejunogastric intussusception has an anatomical or pathological defect, such as a diverticulum or a polyp, been reported as the initiating factor in the telescoping of the bowel. This complication of gastric operation is most readily explained as an accident in the presence of two physiological situations distal to a gastrojejunal stoma. Antiperistalsis normally occurs in the second and third portions of the duodenum, and in humans reverse waves can be seen during the roentgen examination.[1] Obviously, reverse peristalsis must also take place in the efferent limb of a gastrojejunostomy or the disease under discussion could not occur. Secondly, a compensatory dilatation of the jejunum opposite the stoma, and extending for a variable distance into the efferent loop, is a well-recognized roentgen finding and is easily demonstrated at operation, as in the case here reported. These two physiological conditions lend themselves to jejunogastric intussusception. The rarity of this complication attests the accidental nature of its inception; and this explanation is given further credence by the fact that recurrence following surgical reduction was reported in only two instances in the cases reviewed by Bettman,[2] gastroenterostomy having been the initial operation in both cases.

The symptoms and signs of acute jejunogastric intussusception are classical. The characteristic clinical features are sudden onset of colicky, epigastric pain, followed by vomiting and hematemesis in a patient who has had gastric resection or gastroenterostomy. The pain is usually severe and localized. (In the case here reported the pain was poorly controlled by opiates.) The abdomen is soft since the bowel is compromised intraluminally and there is no peritoneal irritation. Tenderness is present on deep palpation in the epigastrium, and a mass may be palpable if the previous operation was gastroenterostomy. After gastric resection, the stomach remnant lies under the left costal margin, and a mass is

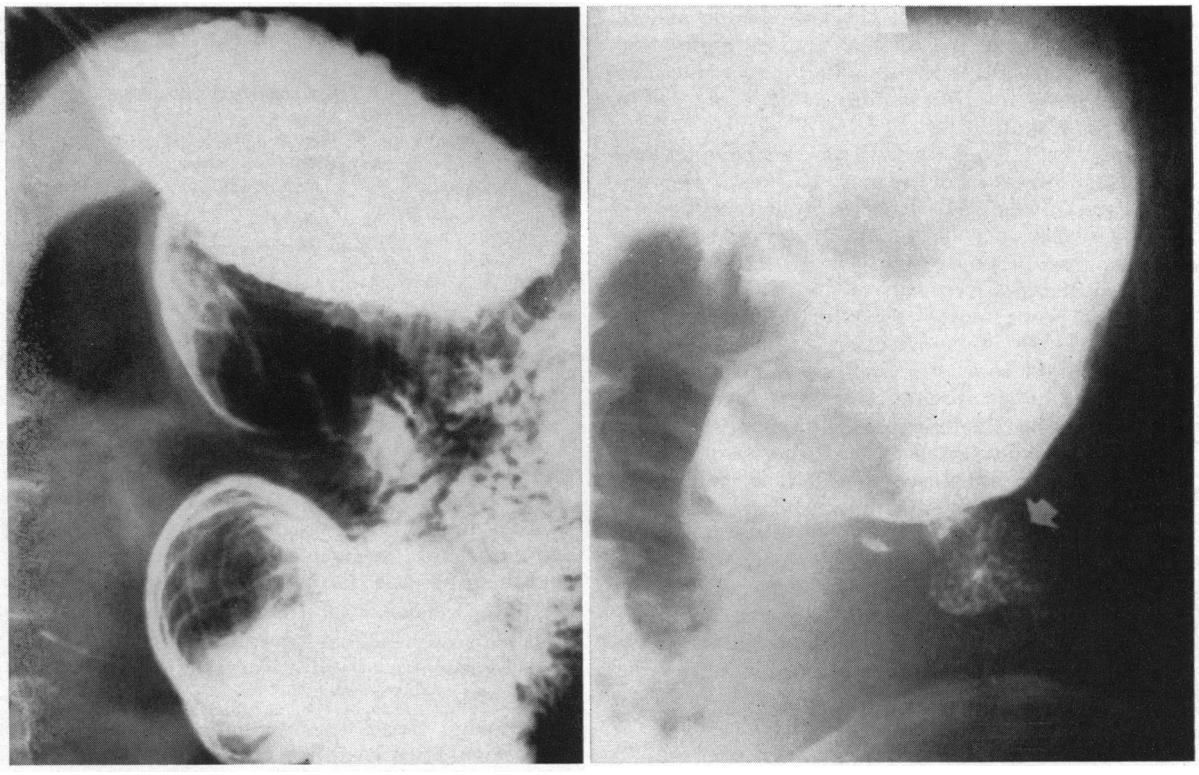

Figure 1.—*Left,* routine barium study of stomach following subtotal gastric resection, taken six months before patient's admission. *Right,* emergency barium study taken shortly after patient's admission with acute jejunogastric intussusception. The stomach remnant is now dilated and contains a filling defect easily recognized as a coil of jejunum. A trickle of barium has entered the afferent loop of the jejunum (arrow).

less likely to be found. In the early stages, peristalsis is normal.

The hematemesis is due to venous engorgement of the trapped loop of intestine. It may not alter the hemoglobin determination significantly. The pathognomonic finding is a filling defect of the stomach, with the characteristics of jejunum, seen after a barium swallow.

The diagnosis rests upon an awareness of this pathological entity, since it is easily confirmed by roentgen examination. In the reported cases of jejunogastric intussusception the usual initial diagnosis was high intestinal obstruction or bleeding marginal ulcer. However, hematemesis is a rare symptom in high obstructions, since bloody bowel content is ordinarily seen in obstruction of the gangrenous, closed-loop type and the vomitus consists of intestinal fluids proximal to this loop. The causes of hematemesis, on the other hand, are rarely accompanied by sudden, colicky pain severe enough to require opiates for relief and suggestive of a bowel obstruction. Of course, the all-important barium examination must be done as an emergency procedure, and must not await the routine schedule "the following morning." Once the scout film of the abdomen has established that the site of the obstruction is not in the colon, no harm is done by ingestion of barium.

The treatment is surgical and it must be prompt

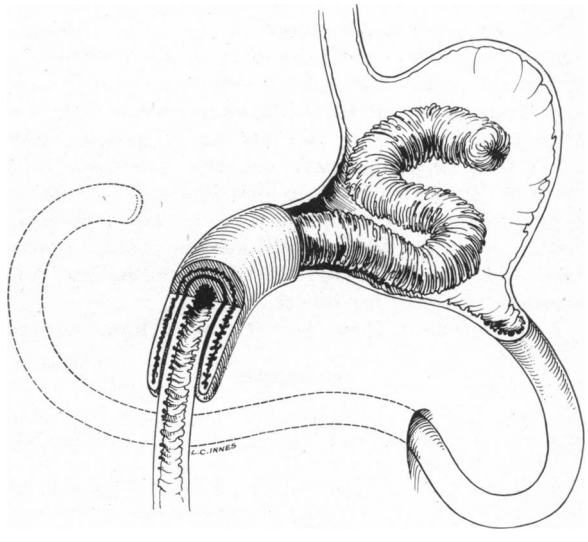

Figure 2.—Diagrammatic sketch of intussusception as seen at operation in present case.

for optimal results. A delay in diagnosis doubly jeopardizes the patient, since additional time will be required to overcome fluid, electrolyte and blood volume deficits incidental to vomiting and gastric suction. This additional delay will often result in gangrenous changes of the intussusceptum, requiring bowel resection, which increases the morbidity and

mortality. At operation the prolapsed loop should be massaged out of the stomach by gentle pressure. In the present case it was impossible to reduce the intussusception by a reasonable amount of traction on the jejunum.

There is a striking similarity between jejunogastric intussusception and the more frequently encountered, and recognized, ileocolic intussusception of infants. Waves of colicky pain are present in both ileocolic and jejunogastric intussusception. The engorged ileum gives rise to blood in the stool ("currant jelly stool") and the prolapse of the jejunum results in hematemesis or the aspiration of bloody fluid by gastric suction. A mass may be palpated in either disease. Roentgen investigation easily establishes the diagnoses, although the barium study cannot be expected to be of therapeutic value in jejunogastric intussusception as it is in ileocolic intussusception. Early diagnosis and treatment of each entity usually eliminates the necessity for bowel resection. Finally, traction on the small bowel during the operative procedure for either disease, in attempting to reduce the intussusception, will lead to disaster. Reduction is accomplished in each case by pressure on the intussusceptum through the walls of the intussuscipiens.

SUMMARY

A case of acute retrograde, jejunogastric intussusception is reported. It is an unusual complication of gastric operations. Without surgical intervention the condition is fatal; unless treatment is prompt, mortality and morbidity rates are high.

Jejunogastric intussusception should be seriously considered in any patient with a previous history of gastroenterostomy or gastric resection who has the usual symptoms of high intestinal obstruction plus hematemesis, with or without an epigastric mass.

As with its counterpart, ileocolic intussusception, barium studies to confirm the diagnosis should be performed on an *emergency* basis. The pathognomonic roentgen findings will lead to prompt operation and, usually, reduction of the intussusception without necessity for bowel resection.

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Dubin-Johnson Syndrome

Report of a Case in a Korean Woman

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In 1954, Dubin and Johnson[4] and Sprinz and Nelson[7] described a new clinicopathological entity characterized by abdominal pain, recurrent jaundice and an unidentified pigment in the liver. The familial nature of the disorder is well documented,[1,8] and the prognosis is uniformly good.

Dubin-Johnson syndrome is distinct from Gilbert's disease, but has several similar features and may represent a quantitative variation of the Rotor syndrome.

Although most cases to date have occurred in Caucasians, two have been reported in Negroes and one in a Korean man.[2,3,6] The purpose of this report is to record a case in a Korean woman.

REPORT OF A CASE

A 29-year-old Korean woman was first seen at the Magan Clinic on May 8, 1961, with complaint of nausea, crampy lower abdominal pain, and diarrhea. In 1958, while the patient was pregnant for the second time, scleral icterus was noted but no abdominal symptoms were present. The pregnancy terminated uneventfully, but two months later nausea, abdominal pain and diarrhea developed abruptly. The diarrhea was annoying and was characterized by approximately five watery but normal colored stools daily. Scleral icterus was again noted by the physician who examined her. In roentgen studies of the upper gastrointestinal tract no abnormalities were noted except for delayed gastric emptying. Cholecystography with the contrast medium ingested was attempted, but the gallbladder was not visualized. A second attempt using twice the usual dosage of contrast medium was also unsuccessful. On January 22, 1959, cholecystectomy and liver biopsy were carried out. The common bile duct was not dilated. The gross liver specimen was described as dark grey-green, and the microscopic sections were interpreted as showing bile stasis. The gallbladder was histologically normal.

Symptoms persisted after operation and the patient consulted another physician. A tentative diag-

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